(FILE 'HOME' ENTERED AT 16:52:30 ON 28 FEB 2002)

FILE 'BIOSIS, EMBASE, MEDLINE, CAPLUS, CANCERLIT' ENTERED AT 16:53:39 ON 28 FEB 2002

```
L1          243 S HCV IRES
                E YAMADA OSAMU/AU
L2          563 S E3
                E YOSHIDA HIROSHI/AU
L3         2162 S E3
                E ZHANG JING/AU
L4          837 S E3
L5         3554 S L2 OR L3 OR L4
L6            1 S L1 AND L5
```

FILE 'CONFSCI' ENTERED AT 16:59:03 ON 28 FEB 2002

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L7            0 S L6
L8            0 S L1 AND L5
```

FILE '1MOBILITY, AGRICOLA, AQUASCI, BIOTECHNO, COMPENDEX, COMPUAB, CONF, CONFSCI, ELCOM, EVENTLINE, HEALSAFE, IMSDRUGCONF, ISMEC, LIFESCI, OCEAN, MEDICONF, PASCAL, PAPERCHEM2, POLLUAB, SOLIDSTATE' ENTERED AT 17:00:36 ON 28 FEB 2002

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L9            0 S L1 AND L5
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d l6 1 ti abs ibib

L6 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS

TI Autogenous Translational Inhibition of Core Protein: Implication for Switch from Translation to RNA Replication in Hepatitis C Virus

AB Pos.-stranded viruses use the genomic RNA as a common template for translation and RNA replication which proceed in inverse direction; a certain regulatory mechanism for translation control is probably required to coordinate these two antagonistic processes. Hepatitis C virus (HCV) core protein is a good candidate that might play a role in such a regulation. In this study, we further investigated whether HCV core protein modulates internal ribosome entry site (IRES)-directed translation. The inclusion of the core-coding sequence significantly suppressed translation initiated by **HCV IRES** in monocistronic and bicistronic reporter systems. The region mainly responsible for this inhibition was mapped to nt 441-473 of the core-coding sequence. This suppression was eliminated by frameshift mutations introduced into this region, suggesting that it is the core protein expressed in cis, rather than the core-coding nucleotide sequence that neg. modulates the efficiency of **HCV IRES** -dependent translation. Furthermore, the core protein provided in trans also specifically decreased the IRES activity in directing cap-independent translation both in transfected cells and in cell-free translation study. Consistently, a gel mobility shift assay showed a specific interaction between the core protein and **HCV IRES**-contg. RNA transcript. These findings suggest that HCV core protein may down-regulate the cap-independent translation as a regulatory mechanism required for initiation of transcription. (c) 2002 Academic Press.

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